Exposure and Vulnerability: Child Health as a Vector in the Intergenerational Transmission of Social Position

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Introduction

How do parents transfer status to their children? Social scientists have emphasized a myriad of mechanisms linking family socioeconomic status (SES) with childhood educational attainment, but children's health merits greater attention. Literature on the origins of poor health and its social consequences is large, but it is also porous. To be sure, poor child health has negative consequences for long term educational attainment, average earnings in adulthood, and occupational status (Case et al. 2005; Haas 2006). Yet, questions remain about the influence of poor child health on educational development in the earliest stages of the life course, and the mechanisms that link poor health with early educational disadvantage.

Using data on children at the earliest observed portion of the life course, this study investigates child health as a mechanism in the intergenerational transmission of educational attainment through an exposure and vulnerability framework. In this framework, exposure refers to the likelihood that parents who are a racial/ethnic minority or socioeconomically disadvantaged will pass on their disadvantage to their children by exposing them to poor health. Vulnerability refers to the extent that exposure to poor child health delays early cognitive development and learning. In addition, this study examines how race and parental investment moderate and mediate the relationship between poor child health and learning. Hypothesizing that race/ethnic minority status will act as a double disadvantage by amplifying the negative influence of poor child health on early learning, and that parental investment will mediate this relationship by accounting for components of family socioeconomic status that influence early

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cognitive development. Through investigating the pathways to poor child health and the subsequent role of early child health on cognitive development and learning in the first years of life, this study emphasizes the importance of child health as a central factor in the intergenerational transmission of social status.

Theoretical Background

The links between family SES, child health, and cognitive outcomes can best be understood through an exposure and vulnerability framework where parents' SES significantly influences child health, and child health, subsequently, has a significant impact on early learning. The first pathway in this framework, e*xposure*, refers to the likelihood that parents, via their social status, expose their child to generally poor health or a particular form of poor health such as low birthweight. Following a social gradient of health perspective, we would expect an inverse relationship between parents' SES and child health where children from high SES positions are least likely to experience poor health while their disadvantaged counterparts are significantly more likely to experience poor health. Indeed, internationally, and within the US, children born to mothers in the lowest social strata are found to have consistently lower health (e.g. low birthweight) than all other strata (Kramer 1987; Spencer 2003; Gorman 1999; Langnase et al. 2002). Similar to SES, disparities in health exposure also persist across race, with blacks suffering a disproportionate amount of child health problems, and white children having the best relative child health (Salsberry and Reagan 2005, Gorman 1999).

The second stage of this framework, v*ulnerability*, refers to the influence of child health on educational attainment. Research in support of the vulnerability association between health and SES in early and late adulthood finds that poor child health has a consistent negative effect on social and educational development in adolescence and adulthood by delaying cognitive development, reducing school readiness, and reducing long term socioeconomic mobility (Aber et al. 1997; Bhutta et al. 2002; Case et al. 2006; Haas 2006; Reichman 2005). Though studies find poor child health to have a negative influence on learning and school performance (Case et al. 2002; Moonie et al. 2006), most research examining health and cognitive development fails to examine the influence of health prior to the start of schooling, effectively unobserving important and formative variations that occur during the first four years of life. Given that the educational skills children begin school with have a dramatic influence on educational attainment in high school and college (Entwisle et al. 2005), health factors that influence cognitive outcomes just after birth are likely to have a critical influence on adult educational attainment, health, and social position.

Double Disadvantage

Decades of social science research suggests that minority race or ethnic status is likely to negatively exacerbate the relationship between health status and a variety of social outcomes (Williams 1990; Moonie et al. 2006). For example, the double jeopardy hypothesis of race and health argues that minority race, by way of discrimination and stigma, amplifies the negative effects of age on poor health as people age along the life course (Ferraro and Farmer 1996). Though evidence exists for a double disadvantage hypothesis later in the life course, the interactive role of race and child health on learning early in the life course is unclear. If, as evidenced in adulthood, race interacts with health to amplify social disparities, it is important to understand how race and health interact in childhood in the formation of educational disparities.

Parental investment

One important way family SES influences the relationship between child health and cognitive development is through parental investment. The parental investment model of educational success posits that parents pass on their economic and educational resources (e.g. family income, IQ, and quality time) to their children to aid in their educational development (Becker 1981; Yeung et al. 2002). While popular notions of parenting suggest that parents equally distribute resources to all of their children, preliminary research suggests that health disadvantaged children receive fewer resources than healthy children within and across families (Datar et al. 2006; Loughran et al. 2004). While the relationship needs greater investigation, part of the correlation between poor child health and delayed cognitive development may be explained by parents investing fewer resources in children with poor health.

Data, Measures, and Analytic Approach

This study uses three waves of data from the Early Childhood Longitudinal Study-Birth Cohort. The ECLS-B is a nationally representative sample, collected by the National Center for Education Statistics, of children born in 2001. The sample used in this analysis is restricted to the 7,468 children with valid responses on the mathematics and literacy assessments in wave three. Wave 1 was collected between 2001 and 2002 when the children were approximately 9 months old. Wave 2 was collected between 2003 and 2004, and wave 3 was collected between 2005 and 2006[2].

Child health. The five measures of child health used include birthweight, asthma, premature birth, APGAR score, and maternal-rated general health. Birthweight is subdivided into three binary variables: normal birthweight (greater than 5.5 pounds), moderate low birthweight (5.5 to 3.3 pounds), and very low birthweight (below 3.3 pounds). Asthma is a binary indicator of ever

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[2] *Additional sociodemographic characteristics*. Socioeconomic status is a constructed continuous standardized composite measure of socioeconomic status of the child using five equally weighted components: father's and mother's education and occupational prestige, and family income (see Ingels et al. 2005 for documentation; single parents use only that parents measures). The five race categories (non-Hispanic White, Black, Hispanic, Asian, and other race) are derived from the mother's report. Additional measures include: gender (binary variable coded as female equal to one), mother's age at the birth the respondent child, whether the child was a twin or multiple birth, number of siblings, whether the mother is married, whether the mother used tobacco or alcohol while pregnant with the respondent child, and a measure of months prior to birth mother sought prenatal care. An age of assessment variable, coded in months, is included in analyses with cognitive dependent variables.

being told by a doctor that the child has asthma. Premature birth is a binary variable coded 1 when childbirth occurs prior to 37 weeks of gestation. The APGAR (Appearance, Pulse, Grimace, Activity, Respiration) score is a positively ranked assessment, ranging from 1-10 (2 possible points for each measure), of child health immediately after childbirth (Apgar 1953). Mother-rated child health includes five categories: (1) excellent, (2) very good, (3) good, (4) fair, and (5) poor.

Cognitive and educational assessments. Waves 1 and 2 measure cognitive development using the Bayley Scale of Infant Development (a measure that asses child development in terms of exploring objects with a purpose, early problem solving, and communicating with words). Wave 3 measures learning using a math (overall mathematics skills) and literacy (reading ability and phonological conventions) assessment (See Mulligan and Flanagan 2006 for greater detail on assessments used).

Parental Investment. Three parental investment measures are used in this analysis including: (1) Was child ever breastfed (yes=1), (2) Months after birth child received well baby care, and (3) a scale of parent and child interaction or HOME scale. The parent-child interaction scale used in this analysis is a composite of five of the parent and child interaction scales (Chronbach alpha=.861) including parental sensitivity, parental stimulation of cognitive development, parental positive regard, child engagement of the parent, and child sustained attention.

Analytic approach

The exposure and vulnerability framework is estimated using OLS and logistic regression. Table 2 investigates the relationship between SES and race on measures of poor child health (exposure). Tables 3 and 4, estimate the influence of child health on Bayley Cognitive scores at waves 1 and 2, and the math and literacy scores at wave 3. In these regressions, Model 1 is a base model including family SES and sociodemographic characteristics, Model 2 adds poor child health as a mediating variable, and Model 3 includes parental investment as a mediator of child health and family SES (vulnerability). Testing the double disadvantage hypothesis, Table 5 five presents predicted values of all 96 race and health interactions.

Findings

Table 1 presents descriptive statistics including means and standard deviations. Results presented in Table 2 support the exposure hypothesis that children born to disadvantaged mothers suffer greater exposure to poor health in early childhood. Looking across models consistent patterns by SES and race emerge. In all but the moderate low birthweight and APGAR models, SES significantly influences child's health, as children from disadvantaged homes are significantly more likely to be born very low birthweight or premature, suffer from asthma, and have lower mother-rated health. In further support of the exposure hypothesis, black mothers are one and half times (odds ratio(or) = 1.568) more likely to birth a moderately low birthweight (MLBW) baby and more than twice (or = 2.191) as likely to birth a very low birthweight (VLBW) baby. Black children are also more likely to be born premature, experience asthma, have a lower APGAR score, and have worse mother-rated health than white children, suggesting racial health disparities are present at the onset of the life course. The child health of whites and Hispanics does not significantly differ. Asian children have significantly better health than whites.

The results in Tables 3 and 4 suggest that at the earliest portion of the life course, children with poor physical health suffer depressed cognitive development and are less prepared for school than their healthy counterparts. In Table 3, family SES is found to be a significant and strong predictor of cognitive development in Model 1. However, controlling for child health in Model 2, the family SES coefficient is reduced 48% at 9 months and 7% at 24 months suggesting child health mediates the relationship between family SES and cognitive development, but that the ameliorative role of child health diminishes as children age. Additionally, being born MLBW or VLBW has a significant negative effect on cognitive development at 9 months. Translating coefficients from model 2 into days ahead or behind comparative groups, we find at nine months MLBW children are 21.2 days behind, and very low birthweight VLBW children are 67.2 days behind normal birthweight children in cognitive development[3]. The penalty of low birthweight increases at 24 months as MLBW children are 28.4 days behind and VLBW children are 110.9 days behind their healthy counterparts. The negative effect of prematurity also increases over time (Wave 1=18.9 days behind; Wave 2=20.5 days behind) shifting premature children further and further behind as they move closer to the start of school. APGAR score, and mother-rated child health are all also significantly associated with cognitive development in expected directions. Model 3 introduces two measures of parental investment as additional mediators at 9 months and a third mediator, HOME score, at 24 months. Though the moderating effect of parental investment is weak at 9 months, parental investment substantially attenuates the relationship between SES, MLBW, VLBW, and mother-rated child health on cognitive development at 24 months. The decrease in coefficient size for child health and SES from 9 to 24 months suggests that the mediating influence of parental investment between SES and child health on cognitive development increases as children age.

The results in Table 4 show that poor child health negatively affects math and literacy skills at the age of four. In model 1, SES is strongly related to math and literacy. However, unlike cognitive development, the introduction of the child health measures does little to explain the influence of SES on math and literacy skills. While APGAR score and asthma are not related

[3] Converting coefficients into days ahead or behind an average student makes for a clearer understanding of the influence on health on learning. In order to calculate a crude estimate of "days behind" I divide the health coefficient by the age of assessment (average learning per month) coefficient and multiply by 30.

to math skills, children with MLBW (39.8 days behind) or VLBW (100 days behind) have significantly delayed math skills compared to normal birth weight children. Children born premature (25 days behind) and children with poor mother-rated child health are less skilled at math at age four. Regressing literacy ability on child health reveals that children with low birthweights (MLBW=32.9 days behind; VLBW=61 days behind), and children born premature (29.2 days behind) have lower literacy skills than healthy children.

Controlling for parental investment in Model 3 reduces the effect of SES and child health on math and reading skills. In particular, the influence of mother-rated health on literacy skills is completely explained by the inclusion of parental investment factors. Though parental investment factors attenuate the effect of MLBW (math=-7%; literacy=-8.6%), VLBW (math=-9%; literacy=-11.6%), and prematurity (math=-11.9%; literacy=-9.7%), all three child health measures remain significant predictors of math and literacy skills. Controlling for parental investment reduces the SES effect on math by 11% and literacy by 8.8 percent. The findings in model 3 in tables 3 and 4 suggests that part of the effect of health on educational success is the result of children who have early poor child health receiving less parental investment. Indeed, poor child health has a significant negative relationship with well baby care, breastfeeding, and the HOME scale (Tables not shown). That birthweight, prematurity, and mother-rated health are all negatively associated with the HOME scale suggests that children with health disadvantages are significantly less likely to receive parental investments.

Taken together, Tables 2, 3, and 4 suggest poor child health has a direct and indirect effect on cognitive development from birth to the start of school. That poor child health is a product of SES, but only attenuates part of the relationship between SES and learning, suggests that part of the influence of SES on cognitive development and math and literacy skills travels indirectly through poor child health. That parental investment mediates part of the relationship between poor child health and the dependent variables, suggests that part of the effect of poor child health on cognitive development and math and literacy skills travels indirectly through parental investment. However, given that the effect of MLBW, VLBW, and prematurity remains large and significant controlling for SES and parental investment indicates that poor child health has a direct effect on cognitive development and math and literacy skills from birth to kindergarten.

The relationship between race and cognitive outcomes is somewhat unexpected. Though blacks fall behind whites in cognitive development in wave 2, in wave 3 blacks are statistically equivalent to whites in math ability, but have more advanced literacy skills. Hispanics, however, are further behind whites in math and literacy skills. Asians, developmentally delayed at the earliest waves, move far ahead of whites in math and literacy skills at wave three. In a test of the double disadvantage hypothesis, Table 5 presents 96 interactions as predicted values in days ahead or behind a white child with similar poor child health. Of the 96 interactions presented, only 8 are significant and they do not appear to follow a consistent pattern. In opposition to the double disadvantage hypothesis, the overall lack of significance in race and health interactions suggests that although disadvantaged groups are more likely to be exposed to poor health, health, once acquired, is an equal opportunity delayer of educational success. Thus, this finding suggests that children of minority race do not experience a double disadvantage in terms of both exposure *and* vulnerability in the relationship between health and education.

Conclusion

This study confirms previous research on the relationship between SES, race and child health outcomes, and expands the literature on the critical role of child health in the intergenerational transmission of educational success in four primary ways. First, using an exposure and vulnerability framework, child health is found to be a central pivot in the relationship between parents SES and early learning because it is sequentially a product of parental SES and then a producer of disparities in early cognitive development, math ability, and literacy. Accordingly, a major contribution of this research is the identification of achievement gaps emerging long before formal schooling. Second, while past research has focused on the negative influence of poor child health on success within school (Conley and Bennett 2000), using ideal data collected at the onset of the life course this study finds evidence for health disparities in cognitive development, math, and literacy skills *just nine months after birth*. Third, expanding on previous evidence of cognitive disparities associated with a single health indicator, these findings identify cognitive disparities resulting from birthweight, prematurity, APGAR score, and Mother-rated child health. Fourth, testing a double disadvantage hypothesis, disparities in cognitive development, math and literacy skills resulting from poor child health are found to *not* vary by race, suggesting that poor child health is an equal opportunity delayer of early learning.

Observing young children at the earliest portion of the life course, this research finds strong evidence of disparities in cognitive development by child health at the start of life. That health at birth has important consequences for early learning suggests that health interventions during gestation and educational support just after birth for children with poor health can have a significant long term effect on life chances. By investigating the pathways to poor child health and the subsequent role of child health on cognitive development and learning in the first years of life, this study provides strong evidence for the importance of child health as a critical factor in the intergenerational transmission of social status.

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Table 1. Descriptive statistics for ECLS-B data (N=7450).

Variable name	Mean	Std. Dev.
Bayley Cognitive Development Score Time 1	75.072	9.888
Bayley Cognitive Development Score Time2	126.246	10.665
Math score	22.617	7.424
Literature Score	13.372	7.109
Moderate low birthweight (< 2500g & >1500g)	.157	
Very low birthweight (<1500g)	.097	
Asthma wave 1	.056	
Premature birth	.272	
Mother-rated child health	1.545	.780
APGAR score	8.777	.863
Family socioeconomic status at Wave 1	.032	.845
Age of assessment in months at Wave 1	10.480	1.857
Age of assessment in months at Wave 2	24.400	1.160
Age of assessment in months at Wave 3	53.002	4.097
White	.458	
Black	.155	
Hispanic	.177	
Asian	.097	
Other race	.112	
Female	.499	
Mother's birth age in years	27.736	6.365
Twin birth	.160	
Number of siblings	1.097	1.135
Mother is married	.789	
Mother used tobacco during pregnancy	.126	
Mother used alcohol while pregnant	.007	
Child was breastfed	.688	
Well baby care (month received)	5.556	2.605
Prenatal care (month received)	2.369	1.360
HOME (Parent-child interaction scale)	22.132	4.170

Table 2. OLS and logistic regression of child health at the onset of life course (N=7450)						
	Dependent variables (independent regressions)					
Independent Variables	Moderate low birth weight	Very low birth weight	Asthma	Premature birth	APGAR	General Health
SES at Wave 1	.961	.853*	.643***	.817***	.029	-.094***
	(.052)	(.056)	(.056)	(.037)	(.016)	(.014)
Black	1.568***	2.191***	2.068***	1.689***	-.163***	.068*
	(.168)	(.253)	(.329)	(.150)	(.043)	(.031)
Hispanic	1.001	.985	1.197	.987	.004	.050
	(.102)	(.118)	(.193)	(.081)	(.028)	(.026)
Asian	.663**	.145***	.720	.483***	.105***	.175***
	(.105)	(.047)	(.215)	(.064)	(.028)	(.031)
Other race	.621***	.534***	1.852***	1.028	-.009	.011
	(.082)	(.090)	(.301)	(.094)	(.034)	(.029)
Female	1.178*	1.205*	.733**	1.032	.001	-.076***
	(.081)	(.096)	(.077)	(.058)	(.020)	(.018)
Mother's birth age	1.001	1.023***	.969**	1.016**	-.004	-.008***
	(.007)	(.008)	(.011)	(.006)	(.002)	(.002)
Twin birth	7.707***	1.828***	.652**	6.232***	-.188***	.031
	(.657)	(.206)	(.105)	(.476)	(.031)	(.027)
Number of siblings	.901***	.837***	1.278***	.945	.026*	.036***
	(.034)	(.040)	(.058)	(.028)	(.012)	(.009)
Mother is married	.926	1.020	.618***	.884	-.039	-.076**
	(.090)	(.111)	(.083)	(.069)	(.032)	(.028)
Tobacco use during pregnancy	2.232***	1.257*	1.237	1.228*	.006	.003
	(.216)	(.149)	(.173)	(.106)	(.031)	(.030)
Alcohol use during pregnancy	.705	1.366	.484	1.078	.057	.117
	(.324)	(.528)	(.350)	(.330)	(.102)	(.131)
Regression type	logit	logit	logit	logit	OLS	OLS

Note: Odds ratios are reported coefficients for logit regressions. Standard errors are in parentheses
† p < .1; * p < .05; ** p < .01; *** p < .001 (two-tailed tests)

Table 3. OLS regression of early cognitive development at 9 and 24 months (N=7450) .

	Bayley Cognitive W1			Bayley Cognitive W2		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
SES at Wave 1	.610***	.316***	.304***	2.913***	2.696***	1.743***
	(.120)	(.104)	(.106)	(.180)	(.174)	(.175)
Moderate low birth weight		-2.858***	-2.839***		-1.817***	-1.677***
		(.208)	(.208)		(.349)	(.338)
Very low birth weight		-9.078***	-8.990***		-7.098***	-6.537***
		(.351)	(.352)		(.494)	(.486)
Asthma wave 1		-.108	-.093		.417	.320
		(.317)	(.316)		(.472)	(.461)
Premature birth		-2.554***	-2.539***		-1.314***	-1.139***
		(.205)	(.205)		(.327)	(.319)
APGAR score		.420***	.416***		.369**	.338**
		(.101)	(.101)		(.143)	(.139)
Mother rated child health		-.415***	-.409***		-.546***	-.387**
		(.093)	(.093)		(.146)	(.141)
Black	-1.276***	.118	.134	-4.303***	-3.345***	-2.658***
	(.285)	(.227)	(.226)	(.382)	(.366)	(.354)
Hispanic	.012	.032	.034	-3.952***	-3.926***	-3.235***
	(.229)	(.193)	(.195)	(.327)	(.314)	(.304)
Asian	-.312	-1.214***	-1.199***	-3.257***	-3.859***	-2.409***
	(.247)	(.228)	(.228)	(.438)	(.436)	(.431)
Other race	.583*	.083	.079	-2.080***	-2.425***	-1.966***
	(.262)	(.230)	(.230)	(.367)	(.356)	(.337)
Female	.442**	.625***	.620	3.328***	3.449***	2.926***
	(.155)	(.131)	(.131)	(.223)	(.216)	(.209)
Mother's birth age	-.050**	-.027*	-.027*	.009	.022	.013
	(.016)	(.013)	(.013)	(.023)	(.022)	(.021)
Twin birth	-3.764***	-1.106***	-1.085***	-3.961***	-2.305***	-2.013***
	(.235)	(.211)	(.211)	(.326)	(.332)	(.322)
Number of siblings	-.139†	-.314***	-.323**	-.704***	-.827***	-.738***
	(.078)	(.066)	(.066)	(.112)	(.106)	(.103)
Mother is married	.458†	.410*	.395*	-.197	-.269	-.467
	(.238)	(.197)	(.198)	(.320)	(.305)	(.297)
Tobacco use during pregnancy	-.885***	-.244	-.236	-.966**	-.571†	-.405
	(.244)	(.199)	(.200)	(.340)	(.325)	(.314)
Alcohol use during pregnancy	-.322	.240	.226	.432	.797	-.066
	(.897)	.691)	(.693)	(1.243)	(1.204)	(1.137)
Timing of prenatal care	.068	-.080	-.083†	.026	-.069	-.068
	(.058)	(.050)	(.050)	(.081)	(.078)	(.075)
Child was breastfed			.091			.372
			(.154)			(.241)
Timing of well baby care			-.066*			-.051
			(.029)			(.040)
HOME score W2						.653***
						(.028)
Age of Assessment	3.785***	4.051***	4.066***	1.891***	1.920***	1.708***
	(.058)	(.057)	(.058)	(.115)	(.115)	(.112)
Constant	37.045	32.547	32.733	1.891	79.484	70.227
R^2	.542	.673	.674	.184	.246	.302

† p < .1; * p < .05; ** p < .01; *** p < .001 (two-tailed tests)

Table 4. OLS regression of early cognitive development at 9 and 24 months (N=7450) .

	Math Score			Literacy Score		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
SES at Wave 1	3.020***	2.940***	2.617***	2.969***	2.919***	2.663***
	(.111)	(.111)	(.114)	(.109)	(.110)	(.114)
Moderate low birth weight		-.831***	-.773***		-.511***	-.467*
		(.227)	(.225)		(.211)	(.212)
Very low birth weight		-2.078***	-1.891***		-.947***	-.837**
		(.308)	(.309)		(.293)	(.294)
Asthma wave 1		-.421	-.447		-.309	-.332
		(.319)	(.316)		(.283)	(.281)
Premature birth		-.522*	-.460*		-.453*	-.409*
		(.204)	(.093)		(.197)	(.197)
APGAR score		.142	.132		-.085	-.091
		(.092)	(.092)		(.088)	(.088)
Mother rated child health		-.150	-.098		-.159†	-.120
		(.094)	(.093)		(.088)	(.088)
Black	-.851***	-.506*	-.274	.529*	.704**	.885***
	(.235)	(.235)	(.234)	(.221)	(.223)	(.224)
Hispanic	-1.513***	-1.510***	-1.329***	-1.289***	-1.284***	-1.168***
	(.206)	(.204)	(.204)	(.190)	(.189)	(.191)
Asian	1.740***	1.534***	1.971***	2.580***	2.494***	2.810***
	(.252)	(.252)	(.254)	(.268)	(.268)	(.269)
Other race	-.769***	-.865***	-.738***	-.159	-.204	-.117
	(.236)	(.235)	(.234)	(.233)	(.233)	(.191)
Female	1.287***	1.321***	1.161***	1.120***	1.131***	1.014***
	(.139)	(.138)	(.137)	(.136)	(.136)	(.233)
Mother's birth age	.106***	.109***	.106***	.099***	.100***	.097***
	(.014)	(.014)	(.014)	(.014)	(.013)	(.013)
Twin birth	-.966***	-.328	-.221	-.170	.224	.304
	(.196)	(.213)	(.212)	(.191)	(.206)	(.206)
Number of siblings	-.827***	-.860***	-.831***	-1.096***	-1.106***	-1.079***
	(.073)	(.073)	(.073)	(.066)	(.066)	(.066)
Mother is married	.582**	.535**	.448*	.348†	.304†	.233
	(.202)	(.200)	(.198)	(.183)	(.184)	(.183)
Tobacco use during pregnancy	-.559**	-.398†	-.314	-.298	-.200	-.121
	(.214)	(.212)	(.209)	(.186)	(.187)	(.186)
Alcohol use during pregnancy	.745	.830	.587	-.767	-.713	-.880
	(.813)	(.808)	(.824)	(.663)	(.681)	(.689)
Timing of prenatal care	-.026	-.058	-.054	-.106*	-.120*	-.115*
	(.053)	(.052)	(.051)	(.050)	(.050)	(.050)
Child was breastfed			.429**			.448**
			(.160)			(.150)
Timing of well baby care			-.024			.005
			(.028)			(.026)
HOME score W2			.195***			.143***
			(.018)			(.018)
Age of Assessment	.620***	.626***	.618***	.462***	.466***	.459***
	(.017)	(.017)	(.017)	(.017)	(.017)	(.017)
Constant	-12.869	-13.826	-17.862	-13.352	-12.292	-15.448
R^2	.350	.363	.375	.320	.324	.331

† $p < .1$; * $p < .05$; ** $p < .01$; *** $p < .001$ (two-tailed tests)

Table 5. Predicted values in days behind for interactions of race and child health on wave 1 and 2 Bayley Cognitive score, and Math and Literacy score at wave 3 (n=7450).

		Poor Child Health Measures					
	Race	Moderate low birth weight	Very low birth weight	Asthma	Premature birth	APGAR	Mother-rated Health
Bayley W1	Black	8.06*	-3.68	7.37	2.12	.96	-2.27
	Hispanic	2.22	4.77	10.47	3.06	2.39	2.73
	Asian	4.36*	-12.90	-2.06	-6.76	-4.86	-3.92
	Other race	-1.64	-6.25	8.37	6.68	.50	-3.95
		Moderate low birth weight	Very low birth weight	Asthma	Premature birth	APGAR	Mother-rated Health
Bayley W2	Black	-62.78	-35.31	-14.09	-44.2	-40.79	-5.31**
	Hispanic	-67.50	-40.44	-72.99	-59.51	-51.96	-33.31
	Asian	-42.74	27.14	-98.2	-38.08	-25.66	-38.81
	Other race	-37.22	-61.23	-18.3	-39.29	-31.2	.47
		Moderate low birth weight	Very low birth weight	Asthma	Premature birth	APGAR	Mother-rated Health
Math Score	Black	-33.65	-6.40	-48.52	1.36	-15.92	26.92
	Hispanic	-42.42	-91.54	-102.23	-48.48	-69.32	19.06**
	Asian	103.81	35.30	-44.39	56.04	107.91	144.53
	Other race	-39.37	-16.05	-52.79	-76.70*	-35.19	-47.82
		Moderate low birth weight	Very low birth weight	Asthma	Premature birth	APGAR	Mother-rated Health
Literacy Score	Black	33.20	57.25	34.24	70.52	52.97	126.13
	Hispanic	-79.67	-152.48*	-38.95	-83.44	-94.94	25.64**
	Asian	148.49	171.50	-6.27	94.22*	222.39	212.40
	Other race	-25.29	-9.80	-49.47	-26.12	7.13	76.09

* $p < .05$; ** $p < .01$; *** $p < .001$ (two-tailed tests)

Note: Each cell represents the interaction between the row race and the column health measure for a given dependent variable. Days ahead or behind in each cell is a measure relative to the predicted values of a white child with a similar child health condition (see text for details on the calculation of days ahead or behind). In theses interactions APGAR is set at 8 and Mother-rate health is set at 4.